RUDOLPH MATAS MEDICAL LIBRARY
TULANE UNIVERSITY
SCHOOL OF MEDICINE

SEP 11 1941

Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION

1790 BROADWAY AT 58TH ST., NEW YORK, N. Y.

DR. JAMES G. CARR, Chicago, *Editor*

DR. GILBERT H. MARQUARDT, Chicago, *Associate Editor*

Vol. X **September, 1941** **No. 9**

THE RELATION OF THE ENDOCRINES TO THE CIRCULATION

The diagnosis of cardiovascular disturbances on an endocrine basis depends on the status of one's knowledge of endocrine diseases.

When abnormal endocrine influences have been acting definitely and over a prolonged period of time, the cardiovascular system may be so damaged that primary cardiovascular disease is thought to be present. And certainly the course of the condition may be like that of a primary cardiovascular disease.

The following is an attempt at a summarized and sketchy correlation of signs and symptoms referred to the circulation as they may occur in conjunction with endocrine disturbances.

Palpitation may well be an expression of the overfunctioning of the thyroid gland. This refers to either regular rhythm or to auricular fibrillation. It is also noted with a paroxysmal rise in blood pressure which may also be associated with a tumor of the adrenal medulla or of the sympathetic chain. Also prolonged medication with Epinephrin, such as typically seen in patients with bronchial asthma, may lead to the same symptom. In all of these instances the heart rate is on the *fast* side. In contrast to this, we note a rather *slow* heart rate with hypothyroidism.

The presence of the syndrome of angina pectoris should make one consider several conditions. When it occurs in women with a normal blood pressure, diabetes should be thought of immediately. It may occur with both hyper- and hypothyroidism, and in both instances, as in diabetes, is probably closely related to coronary artery disease. With reference to thyrotoxicosis, one should not forget the possibility of an intrathoracic goiter. Hypothyroidism is quite rarely associated with angina pectoris but anginal distress may appear under the influence of thyroid medication when the work of the heart is rapidly increased, accompanied by a rise in basal metabolism. In this connection it would be well to issue a warning as to the injudicious use of thyroid extract to combat obesity in middle-aged or elderly patients. One not infrequently observes the development of anginal pain in these cases, and this is certainly an indication to discontinue thyroid medication immediately. Anginal distress occurs characteristically with a paroxysmal rise in blood pressure due to the outpouring of epinephrin into the circulation from a benign tumor of the adrenal medulla or of the sympathetic chain, which is also referred to as pheochromocytoma. Another practical point is the inadvisability of using epinephrin in patients with a history of angina pectoris and cardiac infarction, for instance, in conjunction with local anesthesia. Grave accidents are likely to occur. It is rather generally accepted that attacks of anginal pain provoked by effort, psychic tension or cold are caused by anoxia of the heart muscle. It has been thought that this is closely related to discharges of epinephrin from the suprarenal glands, occurring physiologically under the conditions mentioned. Doctor Raab, of the University of Vermont, has claimed relief of angina pectoris through roentgen irradiation of the suprarenal glands. These attempts are promising and should be further studied.

The mineral and water metabolism is definitely influenced by the endocrines and by endocrine products. This refers, for instance, to the premenstrual retention of sodium, chloride, and water, which may well lead to a transient gain in weight. Unless fully appreciated, the premenstrual retention of fluid in patients suffering from disorders predisposing to fluid accumulation, such as obesity, nephritis, hypoproteinemia or heart disease, could be mistaken for an exacerbation of the underlying disease. In such patients with a tendency to premenstrual fluid retention, benefit is obtained by absolute restriction of sodium chloride in the diet during a seven-to-ten day period preceding onset of menstruation and by the daily administration of 30 cc. of 20% solution of potassium citrate in fruit juice. In patients with mitral stenosis, sinus rhythm, and without evidence of congestion, there may be a tendency to develop pulmonary edema, and in some of these patients the premenstrual period favors this development. The following synthetic hormones have a sodium, chloride, and water retaining effect: the male gonadal hormone, testosterone propionate; the adrenal cortical hormone, desoxycorticosterone ester; and the posterior pituitary hormone, pituitrin. When the male hormone is injected into normal subjects, a gain 1–1.5 kg. in weight is observed within a few days. The adrenal cortical hormone is used in the treatment of Addison's disease, and its effects may be mild-to-severe edema, an increase in the circulating blood volume and in the venous pressure, pulmonary moisture and respiratory distress, particularly if the doses are not kept rather small and if sodium chloride is added to the diet. Pitressin raises the blood pressure and has an anti-diuretic effect, the blood becoming diluted and its volume increased. Therefore, the injection of pituitrin, which contains pitressin, may be damaging to pre-eclamptic patients. There is no objection to the Pitocin fraction. We may add that a rapid gain in weight at the beginning of insulinisation in a diabetic is mostly due to fluid retention.

With regard to the *skin*, in the thyrotoxic patient we note the flushed, warm and moist skin; in hypothyroidism, slight cyanotic discoloration of the face and extremities; in the male human castrate pallor is observed, and it is also noted following an injection of epinephrin or when epinephrin is spontaneously pouring out from a pheochromocytoma, with the accompanying effect of epinephrin from either source—elevation of blood pressure. In hypo-ovarian states, there has been observed redness of the extremities which is sometimes painful and very similar to the picture of so-called erythromelalgia.

A poor *blood pressure* regulation, which sometimes may even lead to vascular collapse, is noted with several hypo-dynamic conditions, namely, in (1) chronic lesions of the adrenal cortex, such as Addison's disease proper, or hemorrhages and degeneration in acute infections such as severe diphtheria; (2) insufficiency of the auterior pituitary lobe and hypogenitalism, two conditions which are not rarely combined. On the other hand, an elevation of the blood pressure is noted in various conditions. The systolic pressure alone may be raised, resulting in a wide pulse pressure, and this is fairly frequently encountered with thyrotoxicosis. An elevation of both the systolic and diastolic pressure is found: (1) with the pituitary-adrenal syndrome of Cushing; (2) in a paroxysmal form with the benign tumors of the adrenal medulla or sympathetic chain, referred to previously and often erroneously diagnosed as anxiety neurosis or hypoglycemia; (3) when hyperparathyroidism has led to nephrolithiasis and subsequently to pyelonephritis; (4) in connection with the injudicious use of the adrenal cortical hormone. It should be added that in females afflicted by fibrous myoma of the uterus a somewhat higher percentage, as compared with control cases, is found to have an elevation of the blood pressure.

Cardiac enlargement is noted in rare instances of eosinophilic tumor of the anterior lobe of the pituitary, leading to the clinical picture of acromegaly. Cardiac enlargement is sometimes erroneously diagnosed in the presence of thyrotoxicosis, the heart being so very hyperactive. Uncomplicated thyrotoxicosis in younger people does not cause cardiac enlargement, but in those with a previously weakened or damaged heart, the constant functional acceleration may well lead to cardiac enlargement and is then usually associated with auricular fibrillation.

Cardiac failure following thyrotoxicosis is of particular importance. It should be clearly realized that a physician may see a patient at a time when certain clinical findings thought to be characteristic for thyrotoxicosis are no longer present. This applies particularly to the thyroid enlargement, exophthalmos, tremor and nervousness. In these cases, basal metabolic readings are not conclusive because (1) basal metabolism is also increased in the presence of congestive heart failure of other origin; (2) the thyrotoxicosis may have burned out and the B.M.R. may have dropped from a previously high level. However, in these patients, several points for diagnosis should be remembered: (1) prior to onset of fluid accumulation they have a history of unexplained weight loss; (2) there is surprisingly long duration of complete disability associated with gross signs of congestive failure; (3) the tachycardia associated with auricular fibrillation does not respond as well as it should to rest and digitalis; (4) a low blood cholesterol level is likely to be present. In elderly females with auricular fibrillation and congestive heart failure, one should suspect the thyrotoxic factor unless there is another obvious etiology.

Roentgenologic examination shows that the filling of the heart is poor in the hypodynamic conditions, particularly in anterior pituitary insufficiency and adrenal cortex insufficiency. The heart in these cases —at least when examined in the upright position— appears quite small, and in Addison's disease there is a rather close correlation between the size of the heart and the severity of the condition. An increase in the silhouette may be observed in connection with myxedema of prolonged duration, but this need not be the case. If an increase in the silhouette is observed, the collection of fluid in the pericardial cavity may be one of the causative factors. Calcifications in the blood vessels are principally noted in two conditions: (1) in diabetes; (2) in hyperparathyroidism, which may be either primary or that which is secondary to kidney disease.

The Electrocardiogram also may show a few findings of interest. It has been mentioned already that auricular fibrillation may accompany thyrotoxicosis. A low voltage E.C.G. is noted in advanced cases of Addison's disease, and there is also a tendency to low voltage, together with flattening and inversion of the T deflections, in connection with myxedema of long duration. Isoelectric or negative T deflections, together with S-T depressions and a lengthening of the Q-T segment, may be noted with hypoglycemia, either spontaneous or induced, and in diabetic acidosis. In this connection it is well to point out that in diabetic patients, who presumably have atherosclerotic heart disease and who receive insulin treatment, hypoglycemia should be avoided. Finally, there is a close relation between the length of the Q-T segment and the amount of physiologically active calcium in the blood serum. A high serum calcium is noted with hyperparathyroidism, causing a shortening of the Q-T segment; while a low serum calcium, as noted with hypoparathyroidism, leads to a prolongation of the Q-T segment.

Hugo Roesler, M.D.
Philadelphia, Pa.